normal in size. Advised medical treatment. This was tried for several months but without avail. Operation decided upon and on November 30th, 1909, nephrotomy was performed. Left kidney exposed and delivered upon back. Slightly lobulated but otherwise normal in appearance. An incision was made along Boerdel's line freely exposing the pelvis. No bleeding points could be seen, although veins of pelvis were slightly congested. Section removed for examination. The kidney wound was then closed, the capsule was turned back, the fatty capsule stitched over kidney, and wound through loin closed except for cigarette drain to kidney. Uneventful recovery with cessation of bleeding. Examination of section removed showed marked arterio sclerosis, glomerulae in various stages of hyaline degeneration, many small areas in which tubules are collapsed and in which there is considerable cellular infiltration and fibrous tissue.

Diagnosis, moderate chronic nephritis. At my earlier report of this case which was before operation, I made a tentative diagnosis of renal varix, but this was disproved and the lesion was shown to be nephritis.

Case 2. Mr. C. C. Consulted me October, 1909. Age 49. Father died of chronic bronchitis, mother of pneumonia.

Personal history: Never sick, except la grippe in 1908. Following this had hernia for which was operated. Denies venereal. Had eruption of foreskin in 1904 for which was treated and recovered in a week. Took no internal medicine.

In July, 1907, noticed urine was bloody. This lasted for a week and was unaccompanied by pain. He knew of no cause for the onset of his trouble unless it was worry. He was in the Charite Hospital, Paris, for seven weeks, during which time he was on a milk diet; took tannin. After leaving the hospital he remained well until February, 1909, when painless hematuria returned. At first it was slight but in a few days became more pronounced and has continued up to the present time. Since February, 1908, has had to arise once or twice at night to urinate, but stream is free and no pain. He is now well nourished; heart normal; lungs normal; abdomen large, palpation negative; penis and testicles normal; prostate normal per rectum; no residual urine.

Cystoscope: Bladder mucosa normal. Ureteral orifices normal. Prostate projects slightly into bladder. Both ureters catheterized. Urinary secretion very free. Urine from left kidney bloody, from right clear. Functional tests, indigo carmin and phloridzin, function normal and both sides equal. Repeated examinations gave parallel findings. Uranalysis. Bladder urine: blood, albumin, no casts, no pus, no epithelium. Right kidney: clear, acid, no albumin, no casts, no pus, no blood. Left kidney: cloudy, acid, albumin present, no casts, many pus cells, many blood cells. X-ray of urinary tract was made. The report by Dr. Cooper says, "There are no abnormal shadows in the region of the kidneys, in the line of the ureters, or in the bladder. The right kidney outline is normal. The left kidney outline is not distinguishable." Moro reaction negative. Wasserman reaction positive. Thinking that the bleeding might come from the pelvis of the kidney, the ureter catheter was introduced into the left kidney pelvis and 5 cc. of adrenalin were injected. It produced no visible effect upon the bleeding. So far in our examination we had eliminated tuberculosis, stone and very probably tumor of any considerable size. The possibility, however, of a slow growing tumor could not be dismissed. The presence of the positive Wasserman indicated a line of therapeutics and the patient was put upon iodide of potash. Two weeks later, Dec. 7th, 1909, the hemorrhage ceased and has not recurred. I believe the cessation of the hemorrhage after the ingestion of the iodide was a coincidence and not the result of the medication, but about this there may be room for a difference of opinion. Last week I cystoscoped the patient again with the following result:

Mixed urine—Albumin none, no casts; few cylindroids; pus, few cells; blood, none.

Right kidney—albumin, none; casts, none; pus, small amount; blood, few cells (probably traumatic).

Left kidney—albumin, none; casts, hyalin and granular; pus, none; blood, none.

The finding of the casts at this examination on the left side would strongly point to the presence of some form of nephritis, probably interstitial.

In the elucidation of this case there are a number of questions that present themselves which time alone can solve. Tumor cannot be absolutely excluded, papillary angioma may be present, angioneurosis is a possibility. After considering all these it is my opinion that we have to deal with a case of hematuria as an initial symptom of chronic nephritis.

THE DIETETIC TREATMENT OF NEPHRITIS.

By RENÉ BINE, M. D., San Francisco.

Since the year 1827, when Richard Bright published his "Report of Medical Cases with a View of Illustrating the Symptoms and Cure of Diseases by a Reference to Morbid Anatomy," until recent years, little was added to our knowledge of the normal and pathological physiology of the kidney, as compared with the progress made in the study of other organs. Therefore, the treatment of nephritis has rested solely upon empiricism. Careful clinical observation, as well as work on experimental nephritis, has lately done much towards explaining the various symptoms of nephritis, and placing its treatment upon a scientific basis.

The diagnosis of nephritis is quite frequently based, primarily upon general symptoms, brought about by impaired renal function. The degree of kidney disturbance can only be estimated by the intensity of these symptoms. On the other hand, such a finding as albuminuria is of lesser importance, for it may be absent in a rapidly fatal case, or be intense in an otherwise healthy person. As soon as the disease process has seriously damaged the eliminating structures of the kidney, certain substances, normally discharged, accumulate in the fluids and tissues of the body. After an increase of urea in the blood of advanced renal cases and of nephrectomized animals had been demonstrated, the term uraemia was applied to the resulting train of symptoms. But the poisonous qualities of urea being afterwards doubted, during the last half century, practically every substance, the elimination of which could be hindered by the kidney lesion, was in turn held responsible. However, of recent years, uraemia was attributed to the presence in the system, of the combination of retained products of metabolism.

In the midst of this darkness, attempts at rational

therapy were made, with the result that milk, and milk alone was earnestly advocated. Meat was objected to by some, because its albuminous molecules were later transformed into urea, and by others because of the possible formation of toxic derivatives in the intestines. Milk was supposed to form less toxic albuminous combinations and derivatives, and thus, it was thought, its beneficial action was explained. On the other hand, it was occasionally found that, if long continued, a milk diet not only proved repulsive to the patient, but that in certain instances produced digestive disturbances, unpleasant general symptoms, emaciation, etc., all of which could be overcome by adding meat to the diet. Nevertheless, there were no established facts to go by, and any change from the classical milk diet was usually made with trepidation.

Recent metabolism experiments have shown that the loss of renal permeability is seldom complete, and that this loss of permeability may be only for certain substances, while the others may be eliminated as in the normal. As a general rule it may be stated that in most cases of acute nephritis, the N-elimination is seriously impaired; only in exceptional cases, occasionally even severe hemorrhagic ones, is it found normal. In chronic cases, the N-elimination varies from time to time, and it can readily be understood that a fatal N-accumulation would soon occur, were the elimination permanently discontinued. In acute nephritis, the kidney is usually unable to get rid of much water, and the anuria is seldom relieved by so-called attempts at flushing out the kidney. In fact, the spontaneous increase in diuresis is the first and surest sign of recovery or else of transition to a chronic course. There are, however, cases of acute nephritis, especially those occurring in the course of acute infectious diseases, where the water-elimination is not interfered with. In chronic parenchymatous nephritis, there is usually a decreased elimination of water; in interstitial nephritis, the reverse is true.

Of particular interest is the Cl-elimination in nephritis, in view of the role ascribed to it in the production of edema. In cases of acute nephritis, at the height of the disease, in severe cases of chronic parenchymatous nephritis, and during the acute exacerbations of, as well as during the periods of failure of cardiac compensation of chronic interstitial nephritis, a marked retention of chlorides occurs. All these conditions are accompanied be edema, or are looked upon as predisposing to edemas. Here, we naturally wish to know whether (1) the water retention be the primary event, either as a result of weak circulation, abnormal permeability of the vessels or of a tissue attraction, salt being secondarily drawn into it to overcome the osmotic pressure of the blood, or whether (2) the retention of Cl be the primary event, the water following into the tissues, according to the law of osmosis. If this retention be primary, is it due to chemical changes in the tissues, as claimed by Acahrd, Loeper, and Laubry, who emphasize the ocurrence of Cl retention in diseases other than nephritis, or is it due, as claimed by Widal, Strauss, etc., purely to renal impermeability? It may be that the water and Cl are simultaneously retained as a result of glomerular injury.

A review of the voluminous literature on the water and salt excretion in nephritis, shows that these questions cannot be positively answered now, but that they, nevertheless, deserve consideration at our hands in the treatment of our cases. For, clinically, we can distinguish symptoms due to Cl retention from those due to N retention.

V. Norden agrees with the French writers, who label as "petite uremie," such conditions as, the high blood pressure, cardiac hypertrophy, headaches, general lassitude, weakness, anorexia, gastro-intestinal disturbances, cachexia, etc., due to the nephritic toxins, instead of reserving the term "uremia" for the state of advanced toxaemia, characterized by clouded mentality, somnolence, convulsions, etc.

Widal has given the name "chlor-uremie" to the symptoms due to a Cl retention. Here we find edemas of the subcutaneous tissues, larynx, serous cavities, as well as the so-called pre-edema, where without any visible watery accumulation in the body, an increase in weight points to its presence. Chloruremie also explains various headaches and eclamptic seizures, as well as certain dyspnoeas, unaccompanied by organic lesions, which disappear after greater or lesser elimination of water and salt. Many instances of severe vomiting and diarrhoea, are attempts of the system at eliminating large amounts of NaCl.

The N-retention and the Cl-retention run an independent course, but at times co-exist. The N-retention is the most dangerous, but here also, much can be done to prevent its occurrence.

From the foregoing, it will be easily seen that every case of nephritis calls for individual study, and that it is impossible to prescribe a diet that will apply, with equal benefit, to all patients with Bright's Disease. It is only after having given this lengthy introduction, that I feel that it will be understood, that I am not laying down any hard and fast rules in a recital of a few generally accepted points in the dietetic treatment of nephritis.

Acute Nephritis. At the beginning and during

the height of an acute nephritis, the most rational treatment is starvation, absolute or relative, according to the severity of the disease. At times, for two or three days, the patient may be allowed but one-half liter of milk, and but sufficient ice to suck, to allay thirst. We know that at this period, water is the most difficult substance for the kidneys to eliminate, and to force a rigid milk or even water diet, is to overload the blood with fluid, and to favor the production of edemas. As soon as the amount of urine has begun to increase, a milk diet may be resorted to. Milk is a food that is easily assimilated, leaving but a small residue, lowering the total of intestinal toxins, and, in proportion to its nutritive value, reducing to the minimum, the digestive work of the gastro-intestinal mucous membranes. In addition, because of its diuretic properties, due to the combined action of its water, salts, and lactose, it is more active and less irritating than any drug that could be prescribed. Furthermore, milk contains on an average of but 1.6 grams of NaCl to the liter, and to this factor is undoubtedly due a great part of its beneficial action, in cases accompanied by edema. But as the fairly large amount of phosphoric acid present in the milk, is eliminated with difficulty by the kidneys, it is better to add to it, a little carbonate of calcium, thus reducing the excretion of phosphorus in the urine.

As the patient improves, it is unwise to rely upon three or four liters of milk, with its 110 to 160 grams of protein producing 30 to 45 grams of urea, for the maintenance of nutrition. It is better not to exceed 1½ liters of milk, adding ⅜ of a liter of cream (375 cc.) to it, thus affording a little over 2000 calories, i. e. with ½ the amount of protein, an amount of nourishment equal to 3¼ liters of good milk.

Gradually, cereals, gruels of oatmeal, barley, sago, rice, and farina, as well as cream cheese, may be added to the diet; likewise, fruits, such as apples, oranges, grapes, and berries, or their juices. With the disappearance of the albuminuria, green vegetables and even meats are given.

In cases where the symptoms of chlor-uremie are in the foreground, even a milk diet affords too great an intake of NaCl, and Widal, Strauss and others, here use a diet still poorer in salt.

Widal includes the following articles in his salt-restricting diet:

Bread: when made without the usual large amount of salt, as patients get along nicely without bread where special baking cannot be obtained.

Meats: (0.5 gr. NaCl. to the pound) served raw, broiled, roasted or boiled, without the addition of salt, but butter, mustard, lemon or a trace of vinegar may be added. Red meats, beef, and mutton are just as good as white meats. Only fresh water fish is permissible.

Eggs: (0.07 Cl per egg) raw or soft-boiled, without any salt. The yolks may also be employed in the sauces used to modify the taste of the various dishes of the menu.

Butter: (0.1 to 1.4% NaCl) if the amount be limited to 50 gr., a maximum of 0.7 gr. of NaCl is given.

Potatoes: boiled, baked, mashed, or in salad form.

Rice: with milk and sugar, or cooked in various forms.

Vegetables: peas (cooked with butter or sugar), carrots, string beans. lettuce, celery, water-cress, artichokes, salads with oil and a small amount of pepper and vinegar are allowed.

All raw fruits, pastry (without salt), jelly, honey, tea, coffee, chocolate, lemonade, syrup, and cider are likewise permissible.

Chronic Parenchymatous Nephritis. Here the diet will necessarily be subject to many variations. In severe cases, the same treatment as in acute nephritis will be beneficial. In a limited number of cases, no diet will avail, whereas, in a third group, the rules applicable to the chronic interstitial form will also apply. It must be remembered that we are dealing with a disease impossible of cure, lasting frequently over a long period, where our aim must be to maintain the general nutrition on as high a plane as possible, and, in so far as compatible with the renal functions, allowing more or less latitude in the diet.

Chronic Interstitial Nephritis. During the acute exacerbations of this disease, the treatment is that described under acute nephritis. In hopeless cases, it is cruel to torture patients with a rigorous diet, and their desires and tastes should be gratified as much as possible. What follows, naturally applies to the average case of contracted kidney.

While we unreservedly condemn the enforcement of a milk diet over a long period of time, we do not mean to exclude it from the diet of chronic cases. In fact, it is good practise to have the patient drink a liter of milk per day. As far as the total amount of fluids is concerned, not more than two liters, and if possible, only 1½ liters should be taken. We well know, that in this disease, sooner or later, cardiac weakness ensues, and it is our duty to postpone this as long as posible. How irrational, therefore, are those, who, actuated by desires to wash out the kidneys, continually impose a strain upon the cardiac muscle. In view of the fact, that a diminished elimination of solid urinary constituents occasionally (and only occasionally) follows a restricted fluid intake, the patient should be permitted to drink, on one day of each week, as much water as he chooses; or else, in the presence of a strong heart, if the patient prefers, he may, once in two or three months, drink two to three liters of fluid per day, for about two weeks. Of

course, weak tea, cocoa, afe au lait, and buttermilk are included in this dietary.

In addition to the above, a mixed diet is to be employed. The nitrogenous food should be distributed throughout the day, being guided in its administration by the symptoms of the patient and by the pulse (i. e. blood pressure). Meats are allowed, usually, once a day. In the past, white meats and fish have been always preferred, but of late, it has been claimed, that red meats were no more harmful than the others. Beef, especially boiled, certainly contains less extractives than the ordinarily prepared white meat. Bacon, ham, tongue, etc., may be given, whereas, brains, liver, kidney, and spleen, should preferably be omitted from the dietary.

Eggs require no special mention. They are classed as a light variety of nitrogenous food. Two or three a day may safely be consumed.

Butter, cream, oil, cereals, and farinaceous foods, gelatins, fruit jellies, and practically every kind of fruit are allowed. Of the vegetables, radishes, horse-radish, and perhaps onions and celery should be forbidden. Asparagus in moderate amounts is not harmful. Condiments, spices, pickles, fried food, rich sauces, meat soups, and meat extracts should certainly be avoided. Sweets should be sparingly eaten, chiefly on account of the danger of digestive disturbances.

In closing, I would warn against trying to make any one man's special diet, fit every case of chronic Bright's. In each individual case, we must feel our way; we must be guided by the nutrition, the state of the patient, all of which are more important than the albuminuria, which always increases on changing from one diet to another, and which at the best, varies but slightly, when one considers, in grams, the amount of albumin eliminated.

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Other references to the experimental side of the question are omitted, being included in the paper dealing particularly with that question.

Discussion.

Dr. G. L. Eaton, San Francisco: In relation to the hemorrhagic conditions found in interstitial nephritis, it is a question whether or not, if these conditions were permitted to exist long enough, there would be a pathological involvement of the kidney. We know that in hypernephroma with hemorrhage long after the nephritis, will be followed by a growth in the kidney. I had a case about eight years ago where there was a hematuria which was periodical about every six months but at the end of eight years it had ceased. Upon careful observation and catheterization and X-ray, a tumor was noticed in the region of the kidney which was found to be some hypernephroma. Was that interstitial condition a forerunner of the hypernephroma? There is no doubt but what infection had a tendency to produce an interstitial nephritis of unilaterial origin. I could state a case which would bear this out.

PELLAGRA; WITH DEMONSTRATION OF A CASE.*

By W. A. CLARK, M. D., San Leandro.

In this paper I shall only endeavor to present very briefly, for those who may not be very familiar with the disease, the salient points of pellagra, and if interested, ask you to take up the time allotted in the examination of this patient, rather than consuming it by giving you what you may obtain as I have done, from the literature.

The disease has been particularly prevalent in Italy, the first description being given in 1735. In 1863 the first cases were reported in the United States.

It is endemic, and is neither contagious, infectious nor inherited. It affects both sexes, any age, and particularly those who labor in the fields.

That damaged corn, as the chief etiological factor in the production of the disease, is asserted and as warmly disputed, it seems probable that the cause of the disease is unknown, except that an unhealthy condition of the mouth and teeth may allow the entrance of the specific poison.

The symptoms are as numerous as inconstant, except the skin manifestations, which are the local expression of a systemic disorder and, in the terminal stage, there is oposthoponos, and occasional temperature.

The prodronal symptoms are languor, general malaise, epigastric pain, diarrhea or constipation, loss of appetite and vertigo. The tongue may become coated or reddened and papillæ prominent, and there may be a loss of membrane, and even ulceration. The papillæ sometimes become the seat of bluish-black pigmentation. The cutaneous lesions usually develop quite suddenly in the early Spring, and occasionally without previous warning. The eruption is usually symmetrical and confined to those portions of the body exposed to the sun. The face and ears may be affected simultaneously and there is usually a collar of pigmentation around the neck, except where the chin shades it. If shoes are not worn, the dorsal surfaces of the feet are affected, missing the heels, giving what is called a "bootleg" appearance. The backs of the hands usually express the typical expression of the disease. The eruption at first is erythematous, spreading from the radial to the ulnar side, the palms being seldom affected. There is usually a sharp line of demarkation at the wrist and the second joint of the fingers. At first the color is dull and red not unlike a sunburn, and indeed I can recollect several of these patients before the disease was called to my attention by Dr. Blue, whom I thought suffered very severely from mere sunburn. The skin is swollen and a burning rather than an itching sensation is felt. The color deepens and does not disappear on pressure, and soon becomes a chocolate or sepia shade. The erythema disappears in about two weeks, with exfoliation. The eruption may become vesicular or even bullous followed by desiccation and crusts. The attack fades, leaving the skin permanently pigmented, thickened, and roughened, a condition readily distinguished at post mortem. The symptoms disap-

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